Resuscitative Hyperkalemia in Noncrush Trauma: A Prospective, Observational Study

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The trauma patient is exposed to physiologic processes and life-saving interventions that predispose to hyperkalemia. Severe elevations in potassium levels subject this compromised patient to additional cardiac risks in the periresuscitative period. Recent advances in the care of the massively traumatized patient may or may not increase the risk for hyperkalemia. This prospective, observational study was undertaken to define the period prevalence of hyperkalemia (plasma potassium level ≥5.5 mmol/L) in a noncrush trauma population during the initial resuscitative period and to identify potential risk factors for the development of hyperkalemia. A total of 131 patients were studied during the initial 12 h after admission for noncrush trauma. The period prevalence of hyperkalemia was 29.0%. Hyperkalemic patients had dramatic shifts in plasma potassium levels compared with nonhyperkalemic patients. Five patients, all from the hyperkalemic group, died. By multivariate logistic regression analysis, independent risk factors for hyperkalemia were an emergency department plasma potassium level of 4.0 mmol/L or higher (relative risk 3.40; 95% confidence interval 1.17 to 9.84; $P = 0.024$ *versus* baseline potassium level <4.0 mmol/L) and transfusion of cell- or plasma-based products (relative risk 10.56; 95% confidence interval 3.62 to 30.78; $P < 0.001$ per log-transformed unit). The prevalence of hyperkalemia during trauma resuscitation was not reported previously. Given the arrhythmic risks of hyperkalemia, particular caution is necessary with trauma patients who present with plasma potassium levels >4.0 mmol/L and require aggressive transfusion support.

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T he deleterious cardiac effects of hyperkalemia have been well described (1). Plasma potassium levels >6.5 mmol/L have been associated with sudden death without preceding evidence of classic, sequential electrocardiographic changes, and there is great interpatient variability in the cardiac response to hyperkalemia (2). Younger patients without renal disease and lower baseline potassium levels may be more susceptible to arrhythmias from mild hyperkalemia and from acute rises in potassium levels (3). In addition, the additive lethality of hyperkalemia in the setting of trauma-related coagulopathy, hypothermia, and acidosis is unknown.

Whereas victims of crush injury commonly develop hyperkalemia as a result of massive release of cellular contents and myoglobinuric tubular toxicity as a direct result of their injury, victims of noncrush, nonburn trauma typically present normo- or mildly hypokalemic, although they subsequently are exposed to a number of interventions during the immediate resuscitative period that have been associated with hyperkalemia in both trauma and nontrauma populations. These interventions include, among others, massive transfusion, aggressive rewarming techniques, and use of neuromuscular blockers perioperatively. Despite this, we are unaware of reports describing the rate of and risk factors for hyperkalemia in the noncrush trauma population.

From observations that have been made during the ongoing military conflicts in Iraq and Afghanistan, the traditional approach to penetrating trauma resuscitation continues to be revised. As a result of an improved understanding of the lethality of trauma coagulopathy, early massive transfusion protocols at combat support hospitals in Iraq for the most severely injured now call for the up-front use of recombinant factor VIIa (NovoSeven; Novo Nordisk, Princeton, NJ); transfusion of plasma in a 1:1 ratio with packed red blood cells (PRBC); and more aggressive use of unmatched, type-specific whole blood for the most severely traumatized patients. Data from animal and civilian studies support this approach (4–6). "Hemostatic," or "damage-control," resuscitation, in which definitive surgical repair is deferred in preference to earlier establishment of hemostatic and metabolic homeostasis, although potentially contributing to the lowest US case-fatality rates in modern warfare, has unknown effects on potassium homeostasis (7,8).

In an attempt to answer these questions, we performed an observational, prospective cohort study at a single combat support hospital in Iraq. Our primary goals were to determine the period prevalence of and risk factors for hyperkalemia in victims of noncrush trauma during the immediate resuscitative period.

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Materials and Methods

The protocol was approved by the combat support hospital's research committee and the investigational review board of Brooke Army Medical Center. The study took place during a 90-d period in 2006. The population consisted of all patients who were admitted to the intensive care unit (ICU) with a primary diagnosis of penetrating, blunt, or explosive trauma. Patients with combined injury mechanisms (*e.g.*, penetrating and blunt) were included and coded according to the primary diagnostic mechanism. Potential subjects were identified at the time of emergency department (ED) arrival. Data were collected from the time of ED evaluation forward for 12 h to include all ED care, operative periods, and intensive care. Censoring events included the end of the 12-h study period and transfer out of the hospital. The primary study end point was the development of hyperkalemia (plasma potassium >5.5 mmol/L), as previously defined (9). Secondary end points were a change in plasma potassium level (defined by the difference between peak and baseline values) ≥1.5 mmol/L and death before the end of the study period.

Exclusion criteria were a primary diagnosis of crush or burn injury, in-hospital death before admission to the ICU, transfer from another health care facility, hyperkalemia or renal failure (plasma creatinine >1.5 mg/dl) at the time of initial evaluation in the ED or known history of chronic kidney disease, and intrahospital transfer from locations other than the ED or operating room (OR) of otherwise qualifying subjects. In addition, patients who initially were identified in the ED as study candidates and ultimately were not admitted to the ICU were excluded from the analysis.

We collected data on qualified subjects during a 12-h period starting with the time of their arrival in the ED. For patients who transferred out of the ICU or who died before completing the 12-h follow-up period, data were collected and analyzed until the occurrence of the censoring event. Variables collected include age and gender, mechanism of injury (penetrating, blunt, or explosive), time from initial injury if available, baseline Glasgow Coma Score, first recorded temperature, type and quantity of all blood products and fluids received, and medications administered. All surgical procedures were documented. In addition, baseline ED values for base deficit and bicarbonate levels from venous blood samples were documented. All plasma potassium levels throughout the study period were recorded. The first recorded plasma creatinine value was reviewed to identify potential exclusion. Blood samples were collected in a standard, "green-top" collection tube that contained lithium heparin. Laboratory analyses were performed using a portable blood gas and electrolyte analyzer (i-STAT Portable Clinical Analyzer; i-STAT Corp., East Windsor, NJ) and a standard laboratory chemistry analyzer (Vitros 250 Chemistry Analyzer; Johnson & Johnson, New Brunswick, NJ). The i-STAT machines were calibrated daily throughout the study period.

Data Analysis

SPSS 12.0 (SPSS, Chicago, IL) was used for all data analyses. For the primary study end point, the highest recorded potassium value during the study period was used. Period prevalence of hyperkalemia was defined as the number of patients who developed hyperkalemia at any time during the study period divided by the total number of study patients. For the secondary end point, a positive outcome was a value ≥1.5 mmol/L when the first recorded potassium value was subtracted from the highest recorded potassium value during the study period. For any independent variable, notation is made in the results section when >10% of values were missing.

The hyperkalemic and nonhyperkalemic groups were compared. In univariate analysis, the independent (two-sample) *t* test and Fisher exact test were used as appropriate. α Values were set at 0.05 (two tailed). The Mann-Whitney test was used as an alternative for the *t* test for continuous variables without Gaussian distribution.

Forward stepwise logistic regression analysis was performed separately to assess factors that were associated independently with the development of hyperkalemia. Variables that were associated at $P < 0.05$ in univariate analysis were included in the logistic regression model for multivariate analysis. However, because of the study sample size, we initially limited the logistic regression model to five covariates (ED potassium, exploratory laparotomy, thoracotomy, ED base deficit, and number of transfused products). Four additional versions of the model were tested using a single additional covariate sequentially (triage directly to OR *versus* ICU, ED bicarbonate, use of a pressor agent, and use of recombinant factor VIIa). The total number of transfused products was skewed rightward and therefore was logarithmically transformed before model entry. Model fitness was assessed by (−2) times the natural log of the likelihood (−2LL); model variance was assessed by the Nagelkerke R^2 coefficient. Hosmer-Lemeshow testing was used to confirm model fitness. Variables that were associated significantly at $P < 0.05$ in multivariate analysis were assumed to be associated independently with the development of hyperkalemia. A receiver operating characteristic curve was developed to assess the predictive accuracy of the logistic regression model.

Results

Of 966 admissions to the hospital from the ED during the study period, 279 were admitted to the ICU. Of these, 148 did not meet criteria for study inclusion. Data were collected and analyzed for the remaining 131 patients. Figure 1 details the flow of patients throughout the study period. Of the 14 patients (nine transfers and five deaths) who did not reach the end of the 12-h study period, existing data until the point of censorship were used in the study analysis. A total of 123 (93.9%) patients had a primary penetrating mechanism of injury. The remaining eight (6.1%) patients were admitted with a primary diagnosis of blunt trauma.

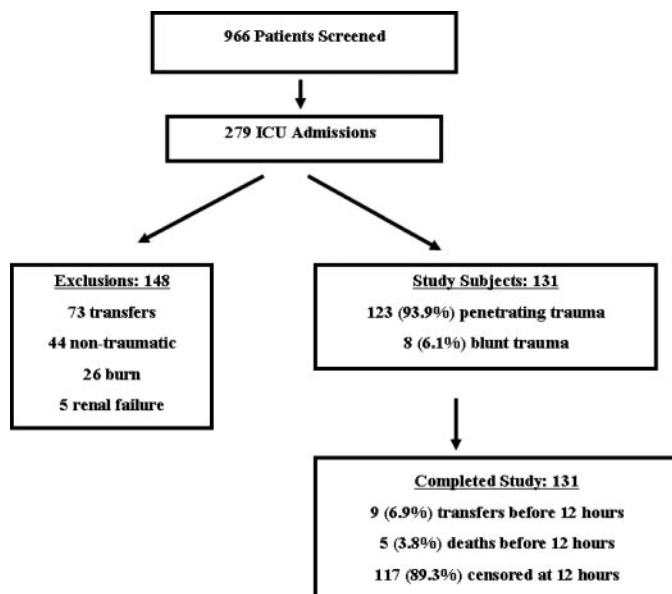Thirty-eight patients developed hyperkalemia sometime dur-

Figure 1. Patient enrollment and disposition.

ing the 12-h follow-up period, for a period prevalence of 29.0%. For the secondary analysis, 50 (38.2%) patients had an increase of ≥1.5 mmol/L in plasma potassium levels during the study period when compared with admission values. When patients with a primary diagnosis of blunt trauma are excluded from the analysis, the period prevalence of hyperkalemia is 30.1% and the incidence of the secondary outcome analysis increases to 39.0%. Five deaths, all in the hyperkalemic group of patients, occurred during the study. There were no deaths in the non-hyperkalemic group.

Figure 2 describes in more detail the baseline and peak plasma potassium levels of the study population. Although more than 97% of the cohort had a baseline, ED potassium level of ≤5.0 mmol/L, more than one third of the population developed a peak potassium level that was higher than this sometime during the study period. Nearly 20% of the population had documented plasma potassium levels >6.0 mmol/L.

The majority of hyperkalemic patients ($n = 29$; 76.3%) first developed hyperkalemia in the OR. Eight (21.1%) and one (2.6%) first became hyperkalemic in the ICU and ED, respectively. In 30 (78.9%) of the 38 hyperkalemic patients, plasma potassium levels normalized before the end of the study period. No patients received diuretic therapy during the study period.

A total of 117 (89.3%) patients remained in the ICU at the end of the 12-h study period. Nine (6.9%) patients were transferred out of the ICU before the end of the study period (mean hours after admission 9.8), and five (3.8%) patients died before the end of the study period.

Table 1 describes the characteristics of, the interventions received by, and the laboratory findings for the entire study population, as well as for the hyperkalemic and nonhyperkalemic subgroups. Results of univariate analysis are included in Table 1. The overall study population consisted predominantly of young men who presented with relatively low potassium values and modest base deficits. The most common procedures performed included exploratory laparotomy, vascular surgery, and orthopedic stabilization and/or repairs. Three fourths of

the overall population received a transfusion; 71.8% of all patients required transfusion of PRBC and/or unmatched, type-specific fresh whole blood. The hyperkalemic group of patients did not differ in age or gender from the nonhyperkalemic group; however, they had significantly higher presenting plasma potassium levels and greater base deficits and were more likely to undergo exploratory laparotomy and thoracotomy. They also were more likely to receive recombinant factor VIIa.

The hyperkalemic group received more transfusions than the nonhyperkalemic group. Of the 38 patients who met criteria for hyperkalemia in our study, 37 (97.4%) received a transfusion of at least one unit of PRBC and/or fresh whole blood. The mean number of units of PRBC transfused in this group was 15.9 (range 0 to 55). Eight (21.1%) of 38 of the hyperkalemic patients received fresh whole blood (mean 2.1 units; range 0 to 22 units). No patient received fresh whole blood without also receiving PRBC.

A total of 123 (93.9%) patients had increases in potassium levels, whereas eight (6.1%) decreased from baseline values. The mean change in plasma potassium level was +1.3 mmol/L. Hyperkalemic patients showed an even more dramatic rise in potassium levels of >2.5 mmol/L over baseline values, compared with modest increases in the nonhyperkalemic group.

Table 2 shows the results of the logistic regression analysis that examined factors that were associated with the development of hyperkalemia. Initial base deficit, exploratory laparotomy, and thoracotomy were not associated with the development of hyperkalemia; ED plasma potassium ≥4.0 mmol/L and transfusion of cell- or plasma-based products both were associated independently with the development of hyperkalemia. The area under the receiver operating characteristic curve for the model was 0.853 (95% confidence interval 0.779 to 0.927; $P < 0.001$; Figure 3). Alternative versions of the model using the identical covariates, with the addition of a single covariate (triage directly to OR *versus* ICU, ED plasma bicarbonate level, use of a pressor agent, or use of recombinant human factor VIIa) did not yield different results and therefore are not reported here.

All patients who died (five of five) met criteria for hyperkalemia, and four of the five were hyperkalemic at the time of death. As a group, these patients had high injury severity scores; required substantial transfusion support; had large base deficits at admission; and experienced acute, substantial increases in plasma potassium levels from baseline. Three of the five had plasma potassium levels >7 mmol/L, and two of the five had levels >9 mmol/L. Table 3 describes in more detail the characteristics of the patients who died.

Discussion

In this population of traumatized patients who underwent aggressive resuscitation, we observed a surprisingly high prevalence of hyperkalemia and identified independent risk factors of ED normokalemia and transfusion of cell- or plasma-based products. Acidosis did not contribute to hyperkalemia in our study, as was reported previously (10).

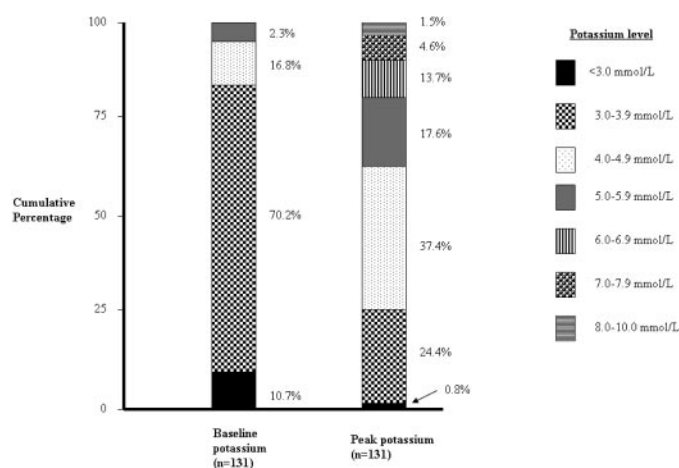Nearly 20% of the population developed potassium levels

Figure 2. Baseline (emergency department [ED]) and peak plasma potassium levels for trauma patients who were admitted to the intensive care unit.

Table 1. Characteristics of study population and univariate analysis of hyperkalemic *versus* nonhyperkalemic subgroups[a]

Variable	Study Cohort ($n = 131$)	Hyperkalemic Patients ($n = 38$)	Nonhyperkalemic Patients ($n = 93$)	P
Male gender (n [%])	123 (93.9)	37 (97.4)	86 (92.5)	0.43
Age (yr)	26.8 ± 8.8	27.8 ± 8.2	26.3 ± 9.0	0.38
Directly to OR (*versus* ICU) from ED (n [%])	109 (83.2)	37 (97.4)	72 (77.4)	0.004
Glasgow coma score >8 (n [%])	109 (83.2)	32 (84.2)	77 (82.8)	1.00
Body temperature (°F)	98.2 ± 1.2	97.9 ± 1.4	98.3 ± 1.2	0.10
ED plasma potassium (mmol/L)	3.7 ± 0.5	4.0 ± 0.7	3.5 ± 0.4	<0.001
Peak plasma potassium (mmol/L)	4.9 ± 1.2	6.5 ± 0.9	4.3 ± 0.6	<0.001
Change in plasma potassium (mmol/L)	1.3 ± 1.2	2.6 ± 1.0	0.7 ± 0.7	<0.001
ED plasma bicarbonate (mmol/L)	23.2 ± 4.0	21.9 ± 4.3	23.6 ± 3.7	0.03
ED base excess (mEq/L)	−4.3 ± 5.2	−6.6 ± 6.4	−3.5 ± 4.4	0.007
Surgical Procedures (n [%])				
exploratory lap	46 (35.1)	20 (52.6)	26 (28.0)	0.009
vascular repair	39 (29.8)	16 (42.1)	23 (24.7)	0.06
ortho procedure	36 (27.5)	11 (28.9)	25 (26.9)	0.83
fasciotomy	21 (16.0)	6 (15.8)	15 (16.1)	1.00
thoracotomy	12 (9.2)	8 (21.1)	4 (4.3)	0.005
amputation	10 (7.6)	2 (5.3)	8 (8.6)	0.72
other	37 (28.2)	3 (7.9)	18 (19.4)	0.12
Recipient of any transfusion product[b] (yes *versus* no; n [%])	99 (75.6)	37 (97.4)	62 (66.7)	<0.001
Transfusion products[c] received (units; median [range])	10.0 (0 to 105.0)	27.5 (0 to 103)	4.0 (0 to 105.0)	<0.001
Recipient of PRBC or whole blood transfusion (yes *versus* no; n [%])	94 (71.8)	37 (97.4)	57 (61.3)	<0.001
Recipient of type-specific, unmatched whole blood[d] (yes *versus* no; n [%])	14 (14.9)	8 (21.1)	6 (6.5)	<0.001
PRBC and/or type-specific, unmatched whole blood received (units; median [range])	5.0 (0 to 72.0)	12.5 (0 to 72.0)	2.0 (0 to 68.0)	<0.001
Succinylcholine (n [%])	86 (65.6)	27 (71.1)	59 (63.4)	0.42
Vecuronium (n [%])	112 (85.5)	36 (94.7)	76 (81.7)	0.06
Recombinant Factor VIIa (n [%])	80 (61.1)	31 (81.6)	49 (52.7)	0.003
Propofol (n [%])	90 (68.7)	28 (73.7)	62 (66.7)	0.53
Ketamine (n [%])	8 (6.1)	2 (5.3)	6 (6.5)	1.00
Etomidate (n [%])	64 (48.9)	20 (52.6)	44 (47.3)	0.70

[a]Normally distributed continuous data are expressed as means ± SD; non-normally distributed data are expressed as the median ± range, as indicated. ED, emergency department; ICU, intensive care unit; lap, laparotomy; OR, operating room; ortho, orthopedic; PRBC, packed red blood cell.
[b]Transfusion products include PRBC; type-specific, unmatched fresh whole blood; platelets; fresh-frozen plasma; or cryoprecipitate.
[c]Expressed as sum of units of PRBC, type-specific unmatched whole blood, and fresh frozen plasma; 10-packs of cryoprecipitate; and six-packs of platelets.
[d]Analysis reflects only the 94 patients who received at least one unit of PRBC or whole blood, not all 131 patients.

Table 2. Multivariate logistic regression model of factors that were associated with the development of hyperkalemia during trauma resuscitation[a]

Covariates[b]	RR for Hyperkalemia (95% CI)	P
ED potassium ≥4.0 (*versus* <4.0 mmol/L)	3.39 (1.18 to 9.84)	0.02
Exploratory lap (yes *versus* no)	1.76 (0.62 to 4.95)	0.29
Thoracotomy (yes *versus* no)	2.10 (0.43 to 10.33)	0.36
ED base deficit <−9 (*versus* ≥−9 mEq/L)	1.55 (0.31 to 7.70)	0.59
Total transfused products (per log-transformed unit)[c]	10.56 (3.62 to 30.78)	<0.001

[a]CI, confidence interval; RR, relative risk.
[b]Four alternative versions of the logistic regression model were obtained using the above five covariates with one of four additional covariates added sequentially (triage directly to OR *versus* ICU, ED plasma bicarbonate, use of a pressor agent, and use of recombinant human factor VIIa). The identified risk factors and their associated RR remained unchanged in these alternative versions of the model.
[c]Transfused products include PRBC; type-specific, unmatched fresh whole blood; platelets; fresh-frozen plasma; and cryoprecipitate.
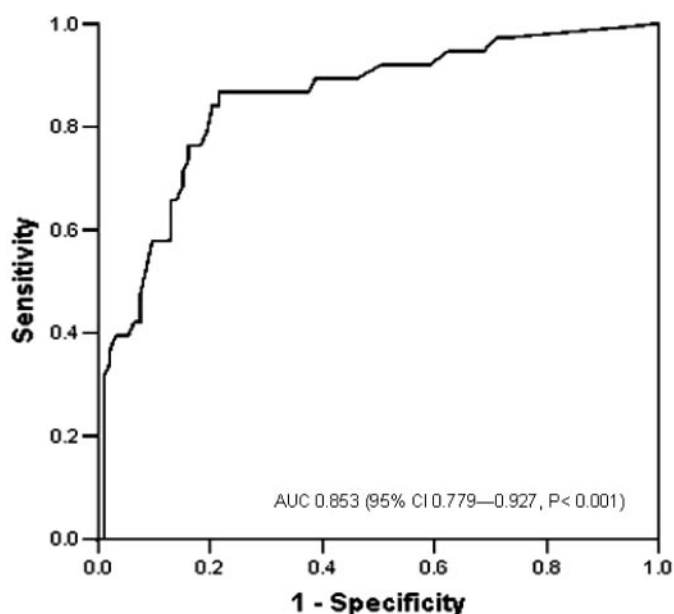
Figure 3. Receiver operating characteristic curve of logistic regression model with baseline (ED) plasma potassium and total transfused products as covariates.

>6.0 mmol/L, at which the risk for cardiac arrhythmias rises substantially. In addition, marked acute rises in potassium from baseline admission values occurred in a substantial proportion of the population during a period of hours, heightening the importance of identifying at-risk patients early to optimize therapies that are designed to ameliorate the impact of elevated potassium levels. Death occurred only in the hyperkalemic group; potassium levels >6.5 were observed in four of these five patients.

We are unaware of prospective data on the prevalence of hyperkalemia in trauma populations. As points of comparison, in unselected populations of hospitalized patients, retrospective analyses have reported the prevalence of hyperkalemia to be between 1.7 and 5.2% (11–13). In victims of the crush syndrome, hyperkalemia as a result of cellular breakdown and massive intracellular release is a common electrolyte abnormality and a common cause of death in initial survivors (14). In the most extensively reported disaster that produced large numbers of crush syndrome casualties, the Marmara earthquake in Turkey in 1999, Erek *et al.* (15) reported an incidence of hyperkalemia of 42% in victims who survived to hospitalization. No specific criterion for hyperkalemia was included in their report, however, making comparisons with our group problematic. In addition, their population consisted of patients who were transported to hospitals with hemodialysis capabilities; the true incidence of hyperkalemia in populations of crush patients who survive to hospitalization therefore may be lower.

Fresh whole blood, plasma-poor PRBC, and fresh plasma all contain exogenous potassium. These products represent the vast majority of transfusions (with the remainder composed of platelets and cryoprecipitate) that were given to the patients in our study. Although we did not routinely measure the potas-

sium concentration from our stored or fresh blood products, the exogenous potassium load from a unit of PRBC is thought to increase as shelf life increases; because of the need to store continuously massive quantities of PRBC and challenges in shipping from US-based sources to locations within the theater of operations in Iraq, the average shelf life of a unit of PRBC is between 30 and 35 d at our hospital. Brown *et al.* (16) showed that the amount of potassium that is contained in the small plasma component of a stored unit of PRBC seems to plateau at approximately 20 d and averages >3.7 mmol at this time, representing a clinically significant source of exogenous potassium if multiple units are given. However, as these authors pointed out, there is wide variability in the plasma potassium concentration of stored PRBC, independent of shelf life. Fresh whole-blood transfusion for the massively traumatized patient who requires large-volume transfusion has theoretical hemostatic advantages, despite the potential risks, and is an integral part of some military and civilian trauma resuscitation programs; whether there are clinical advantages to the use of whole blood over PRBC with respect to potassium homeostasis awaits further study.

Published reports conflict with respect to the relationship between massive blood transfusion and hyperkalemia. A review by Wenze (17) in 1993 noted the occurrence of hyperkalemia to be uncommon after massive blood transfusion. Rudolph and Boyd (18), in a review that was published in 1990, commented that hyperkalemia is less likely to occur secondary to transfusion than to underresuscitation. Other reviewers have noted this complication (19). In the only prospective trial of which we are aware, Parshuram *et al.* (20) reported on 28 critically ill pediatric patients who underwent transfusion with PRBC. Hyperkalemia was not observed. Linko *et al.* (21), in a retrospective analysis of 21 patients who underwent transfusion of 10 or more units of whole blood, identified transient hyperkalemia (potassium values >6.0 mmol/L) in approximately 50% of the patients during the period of active transfusion; however, hyperkalemia correlated with the transfusion rate and not the total amount of transfused blood. Parshuram's group also investigated the role of transfusion rate in their prospective analysis, and although they concluded that this had no effect on the development of hyperkalemia, only six of their 28 patients received bolus transfusions.

Brown *et al.* (22), in turn, reported retrospectively on 138 pediatric patients (combined medical and trauma patients) who sustained cardiac arrest and compared patients who received a rapid blood transfusion with those who did not. During cardiac arrest, the mean plasma potassium concentration in the nontransfused group *versus* the transfused group was 5.6 *versus* 8.2 mmol/L, which was statistically and clinically significant. The authors hypothesized, on the basis of these findings and an associated simulation that was developed to mimic hypovolemic pediatric cardiac arrest, that the combination of a low cardiac output state and an acute potassium load from rapid blood transfusion predisposed patients to hyperkalemia. This analysis has direct implications for the findings reported herein: The patients in our analysis presented with severe trauma

Table 3. Deaths[a]

Age	Gender	Injury	Surgical Procedures	ISS	Cause of Death	Initial Base Excess (mEq/L)	Total Products Transfused (Units)	Baseline Potassium (mmol/L)	Highest Potassium (mmol/L)	Last Potassium before Death (mmol/L)
11 yr	M	Shrapnel to abdomen	Exploratory lap, vascular repair	29	Exsanguination	−9	90	2.9	6.7	6.7
25 yr	M	Shrapnel to abdomen, lower extremity	Completion of amputation, two exploratory lap	50	Exsanguination	−28	54	5.3	7.3	6.7
2 mo	M	Shrapnel to abdomen, lower extremity	Exploratory lap, completion of amputation	50	Penetrating wounds	Not obtained	5	5.3	9.2	9.0
22 yr	M	Shrapnel to abdomen	Exploratory lap, ortho repair	75	Traumatic brain injury	−16	72	4.8	6.2	5.3
27 yr	M	Gunshot wounds to abdomen and chest	Exploratory lap, thoracotomy	29	Penetrating wounds	0	74	3.2	9.1	9.1

[a]ISS, injury severity score (23).

and baseline hypovolemia as a result of active hemorrhage and volume depletion before admission (as evidenced by substantial base deficits at the time of presentation to the ED). The majority of patients in our study received bolus product transfusion *via* access directly into the central circulation.

Our study has limitations. The true prevalence of hyperkalemia in hospitalized trauma patients may be different because only patients who were admitted to the ICU were included in this analysis. Although the majority of patients sustained penetrating trauma, it is likely that many sustained combined mechanisms of injury, to include blunt and/or explosive injuries along with penetrating mechanisms; this would not be accounted for with the method that we used. Finally, although our hospital routinely uses a rapid blood transfuser in both the OR and the ICU, we did not systematically track transfusion rates during our study and therefore cannot assess the impact that this may have had on our observations.

Conclusion

The period prevalence of hyperkalemia in this population of primarily penetrating trauma patients is surprisingly high, and we have identified objective risk factors of ED normokalemia (plasma potassium >4.0 mmol/L) and transfusion of cell- and/or non–cell-based transfusion products. We believe the predictive accuracy of our model allows for the identification of at-risk patients and the timely implementation of appropriate interventions to address elevated plasma potassium levels. The concept of prophylactic management of high-risk patients is not addressed in this observational study; large-scale, preinfusion washing of cell-based transfusion products for the massively traumatized patient, particularly in a mass-casualty situation, would be impractical. Likewise, the impact of patient-directed therapies to reduce the extracellular potassium load in at-risk, traumatized patients, such as the use of insulin, resin binders, or loop diuretics, is unknown and cannot be recommended without further study for a population of trauma patients who undergo active resuscitation. The use of fresh whole blood and PRBC with limited storage time may reduce the burden of hyperkalemia, although studies are needed to test this hypothesis. On the basis of the findings reported here, our practice has changed to increase the frequency of labo-

ratory testing of high-risk patients to identify rising potassium levels earlier and thereby allow for earlier intervention.

Disclosures

None.

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